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Issue Date: 06 June 2003

In the Matter of

Edd Logan,
Claimant

v.

Clinchfield Coal Company and Acordia Employers Service,
Employer/Carrier

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

Case No. 2002-BLA-390

DECISION AND ORDER DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs, for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising form coal mine employment and is commonly known as "black lung."

A formal hearing took place before the undersigned on February 6, 2003 in Abingdon, Virginia. At that time, I admitted Director's Exhibits 1 through 48, Claimant's Exhibits 1 through 9, and Employer's Exhibits 1 through 39. I allowed the Employer additional time to submit exhibits after the hearing, which were subsequently admitted as Employer's Exhibits 40 and 41. The Employer submitted a brief on May 23, 2003; neither the Claimant nor the Director submitted a brief.

I have based my analysis on the entire record, including the exhibits and representations of the parties, and have given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

JURISDICTION AND PROCEDURAL HISTORY

The Claimant, Edd Logan, filed a claim for benefits on October 25, 1999 (DX 1). The District Director denied the claim on February 2, 2000 (DX 15), and the Claimant requested a hearing (DX 16). Instead, the Director held an Informal Conference, where the Claimant's claim was denied. The Claimant submitted additional medical evidence, and on March 13, 2002, the Director issued a Proposed Decision and Order Denying Request for Modification (DX 41). On April 13, 2002, the Claimant appealed, and requested a formal hearing before an Administrative Law Judge (DX 43).

The Claimant filed an earlier claim on August 22, 1980, which was denied by the Director on June 4, 1981 (DX 44-5). The Claimant did not further pursue that claim. On September 1, 1982, the Claimant filed a second application for benefits, which was denied by the Director on September 26, 1983 (DX 45-22). The Claimant requested a formal hearing, which was held before Administrative Law Judge V. M. McElroy (DX 45-46). Judge McElroy issued his Decision and Order denying benefits on February 23, 1987 (DX 45-48). Claimant appealed to the Benefits Review Board (Board), which issued a Decision and Order affirming Judge McElroy's denial of benefits on February 23, 1987 (DX 45-52).

The Claimant filed another application for benefits on March 24, 1989, and the Director issued a Proposed Decision and Order of No Material Change in Condition on June 22, 1989 (DX 45-62). The Claimant requested that his application be held in abeyance pending a decision by the Board in another proceeding (DX 45-65). The Claimant was notified that his claim was being denied on December 7, 1992, and he requested a formal hearing, which was held before Administrative Law Judge Robert D. Kaplan (DX 46-15, 46-32). Judge Kaplan issued his Decision and Order Denying Benefits on February 23, 1994 (DX 46-35).

By letter dated February 2, 1995, the Claimant submitted a report from Dr. Myers, and requested modification (DX 46-35). On August 21, 1995, the Director issued a Proposed Decision and Order Denying Request for Modification (DX 46-48). The claim was referred to the Office of Administrative Law Judges, and a hearing was scheduled before Administrative Law Judge Thomas F. Phalen Jr. on February 25, 1997 (DX 46-57). At that time, the Claimant's representative requested that the Claimant be allowed to withdraw his claim, so that he could have time to further develop his evidence. On April 21, 1997, Judge Phalen issued a formal Order of Dismissal (DX 46-58).

ISSUES

The issues in this case are:

1. Whether the Claimant has pneumoconiosis;

2. If so, whether the Claimant's pneumoconiosis arose out of his coal mine employment;

3. Whether the Claimant has a total respiratory or pulmonary disability; and

4. If so, whether the Claimant's total respiratory or pulmonary disability is due to pneumoconiosis.

(DX 47).

APPLICABLE STANDARD

As the Claimant's October 25, 1999 claim was filed more than one year after his earlier claim was finally denied by Judge Kaplan, it is considered a duplicate claim and must be denied pursuant to 20 C.F.R. Section 725.309 unless the Claimant can show that there has been a material change in conditions since the date of denial of the prior claim.[1] If the Claimant is successful in showing such a change, then his claim must be evaluated under Part 718. *Dotson v. Director, OWCP,* 14 B.L.R. 1-10 (1990).

In *LaBelle Processing Co. v. Swarrow,* 72 F.3d 308 (3rd Cir. 1995), the Third Circuit adopted the Sixth Circuit's standard for finding a "material change in conditions:"

> [T]o assess whether a material change is established, the ALJ must consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proven at least one of the elements of entitlement previously adjudicated against him. If the miner establishes the existence of that element, he has demonstrated, as a matter of law, a material change. Then the ALJ must consider whether all of the record evidence, including that submitted with the previous claims, supports a finding of entitlement to benefits.

Sharondale Corp. v. Ross, 42 F.3d 993, 997-998 (6th Cir. 1994). This standard was also adopted by the Fourth Circuit in *Lisa Lee Mines v. Director, OWCP,* 57 F.3d 402 (1995) *aff'd*., 86 F.3d 1348 (4th Cir. 1996)(*en banc*).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

[1] Contrary to the Employer's argument, this is a duplicate claim only; it is not a request for modification of the Director's denial, which was not a final determination. Thus, the operative date for demonstrating a material change in conditions is February 23, 1994, the date of Judge Kaplan's denial, not February 5, 2001, the date of the Informal Conference.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted and arguments made.

Background

The Claimant, Edd Logan, was born on March 31, 1924 (DX 1). He married his wife, Alene West, on December 12, 1947, and they reside together (DX 1, 8). The Claimant has no children who are under 18 or dependent upon him (DX 1). I find that the Claimant has one dependent for purposes of augmentation of benefits.

Based on a stipulation by the parties, Judge Kaplan found that the Claimant worked in coal mine employment for at least thirty three years. In his 1987 Decision, Judge McElroy also found that the Claimant had thirty three years of coal mine employment. These determinations are amply supported by the record, and I find that the Claimant has thirty three years of coal mine employment. The Employer does not contest its status as the responsible operator, and the record reflects that the Claimant worked for over sixteen years for the Employer, ending in 1983. I find that the Employer is properly named as the responsible operator.

Newly Submitted Medical Evidence

The following new medical evidence is in the record.

X-ray Evidence[2]

Exhibit No.	***Date of X-ray***	***Reading Date***	***Physician/ Qualifications***	***Impression***
DX 46-38	1-12-95	2-9-95	Bassali/B, BCR	1/1, p, s
DX 46-35	1-12-95	1-12-95	Myers	1/1, p, s
DX 46-36	1-12-95	3-6-95	Sargent/B	Negative for pneumoconiosis
DX 46-42	1-12-95	6-23-95	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-42	1-12-95	6-20-95	Spitz/B, BCR	Negative for pneumoconiosis
DX 46-51	1-12-95	8-23-95	Scott/B, BCR	Negative for pneumoconiosis

[2] B-B reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
DX 46-51	1-12-95	8-25-95	Wheeler/B, BCR	Negative for pneumoconiosis
DX 46-53	1-12-95	10-26-95	Fino/B	Negative for pneumoconiosis
DX 46-35	2-22-95	2-24-95	Bushey	2/1, p, q
DX 46-38	2-22-95	3-9-95	Bassali/B, BCR	1/1, q, t
DX 46-40	2-22-95	3-28-95	Sargent/B	Negative for pneumoconiosis
DX 46-55	2-22-95	2-28-96	Spitz/B, BCR	Negative for pneumoconiosis
DX 46-46	2-22-95	8-14-95	Barrett/B, BCR	Negative for pneumoconiosis
DX 46-55	2-22-95	2-27-96	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-55	2-22-95	3-29-96	Fino/B	Negative for pneumoconiosis
DX 46-55	5-15-95	4-3-96	Spitz/B, BCR	Negative for pneumoconiosis
DX 46-55	5-15-95	4-4-96	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-55	5-15-95	3-29-96	Fino/B	Negative for pneumoconiosis
DX 46-41	5-22-95	5-22-95	Vuskovich/B	Negative for pneumoconiosis
DX 46-50	5-22-95	8-19-95	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-43	5-22-95	7-2-95	Pendergrass/B, BCR	Negative for pneumoconiosis
DX 46-50	5-22-95	8-14-95	Spitz/B, BCR	Negative for pneumoconiosis
DX 46-44	7-25-95	7-25-95	Dahhan/B	Negative for pneumoconiosis
DX 46-50	7-25-95	8-19-95	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-50	7-25-95	8-14-95	Spitz/B, BCR	Negative for pneumoconiosis
DX 46-52	7-25-95	9-29-95	Pendergrass/B, BCR	Negative for pneumoconiosis
DX 46-55	6-20-96	6-20-96	Sargent/B	0/0
DX 46-55	6-20-96	9-26-96	Wheeler/B, BCR	Negative for pneumoconiosis
DX 46-55	6-20-96	7-3-96	Wiot/B, BCR	Negative for pneumoconiosis
DX 46-55	6-20-96	7-15-96	Spitz/B, BCR	Negative for pneumoconiosis
DX 13	1-6-00	1-6-00	Paranthaman/B	Negative for pneumoconiosis
DX 14	1-6-00	1-21-00	Barrett/B, BCR	Negative for pneumoconiosis

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
DX 23	1-6-00	5-15-00	Wheeler/B, BCR	Negative for pneumoconiosis
DX 23	1-6-00	5-15-00	Scott/B, BCR	Negative for pneumoconiosis
DX 24	1-27-00	1-27-00	Ramakrishnan/B, BCR	0/1, p, s
DX 33	1-27-00	11-9-00	Fino/B	Negative for pneumoconiosis
DX 33	1-27-00	10-21-00	Wheeler/B, BCR	Negative for pneumoconiosis
DX 33	1-27-00	10-20-00	Scott/B, BCR	Negative for pneumoconiosis
DX 24	2-3-00	2-4-00	Ramakrishnan/B, BCR	0/1, p, s
DX 27	2-3-00	3-4-00	Alexander/B, BCR	½, p, q
DX 33	2-3-00	11-9-00	Fino/B	Negative for pneumoconiosis
DX 33	2-3-00	10-21-00	Wheeler/B, BCR	Negative for pneumoconiosis
DX 33	2-3-00	10-20-00	Scott/B, BCR	Negative for pneumoconiosis
DX 35	2-3-00	1-20-00	Barrett/B, BCR	Negative for pneumoconiosis
DX 24	5-8-00	5-8-00	Ramakrishnan/B, BCR	0/0
DX 33	5-8-00	10-20-00	Scott/B, BCR	Negative for pneumoconiosis
DX 33	5-8-00	11-9-00	Fino/B	Negative for pneumoconiosis
DX 33	5-8-00	10-21-00	Wheeler/B, BCR	Negative for pneumoconiosis
DX 26	9-6-00	9-11-00	Fino/B	Negative for pneumoconiosis
DX 29	9-6-00	9-18-00	Scott/B, BCR	Negative for pneumoconiosis
DX 29	9-6-00	9-15-00	Wheeler/B, BCR	Negative for pneumoconiosis
DX 33	9-6-00	10-26-00	Binns/B, BCR	Negative for pneumoconiosis
DX 36	11-7-00	11-7-00	Patel/B, BCR	1/1, s, s
EX 1	11-7-00	6-3-02	Scatarige/B, BCR	Negative for pneumoconiosis
EX 2	11-7-00	6-3-02	Scott/B, BCR	Negative for pneumoconiosis
EX 36	11-7-00	10-3-02	West/B	Negative for pneumoconiosis
EX 3	12-26-00	6-3-02	Scatarige/B, BCR	Negative for pneumoconiosis

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
EX 4	12-26-00	6-3-02	Scott/B, BCR	Negative for pneumoconiosis
EX 35	3-15-01	10-3-02	Fino/B	Negative for pneumoconiosis
EX 5	3-15-01	6-3-02	Scott/B, BCR	Negative for pneumoconiosis
EX 6	3-15-01	6-3-02	Scatarige/B, BCR	Negative for pneumoconiosis
DX 36	10-17-01	10-17-01	Smiddy	Micronodular changes in all six lobes consistent with previous diagnosis of pneumoconiosis
DX 40	10-17-01	3-4-02	Barrett/B, BCR	Negative for pneumoconiosis
EX 10	10-17-01	8-23-02	Scatarige/B, BCR	Negative for pneumoconiosis
EX 9	10-17-01	8-23-02	Wheeler/B, BCR	Negative for pneumoconiosis
DX 42	2-27-02	2-27-02	Dahhan/B	Negative for pneumoconiosis
EX 34	2-27-02	10-3-02	Fino/B	Negative for pneumoconiosis
EX 7	2-2-7-02	6-3-02	Scatarige/B, BCR	Negative for pneumoconiosis
EX 8	2-27-02	6-3-02	Scott/B, BCR	Negative for pneumoconiosis
EX 37	12-17-02	1-6-03	Wheeler/B, BCR	Negative for pneumoconiosis
EX 38	12-17-02	1-7-03	Scott/B, BCR	Negative for pneumoconiosis
EX 39	12-17-02	12-17-02	Hippensteel/B	0/1, s, p

Pulmonary Function Studies

Exhibit No.	*Date*	*Age/Ht*	*FEV1*	*FVC*	*MVV*	*Effort*
DX 46-35	1-12-95	70/74"	2.33 2.5	3.91 4.13	75.5 75	Good
DX 46-35	2-22-95	70/74.5"	2.6	4.2		
DX 46-41	5-22-95	71/74"	2.26 2.69	4.37 4.7		Good
DX 46-44	7-25-95	71/74"	2.34 2.62	4.54 4.67	88.93 84.5	Fair

DX 46-56	4-2-96	72/75"	2.32 2.55	4.29 4.42		Good
DX 46-55	6-20-96	72/74"	2.53 2.83	5.03 5.02	74	Good
DX 27	8-30-99	75/75"	1.71	3.24		Good
DX 9	1-6-00	75/73"	2.08 2.04	4.01 4.4	100 92	Good
DX 27	1-27-00	75/76"	1.79 1.95	3.3 3.83	62 72	Good
DX 31	7-26-00	76/75"	1.72	3.43		Good
DX 26	9-6-00	76/73"	1.76 1.78	3.91 3.94	60	Good
DX 36	11-7-00	76/73"	1.79 1.99	4.6 4.94	64	
DX 36	7-25-01	77/75"	1.81	3.48	71	Good
DX 36	10-17-01	77/75"	2.12	4.86		Good
DX 42	2-27-02	77/74"	1.62 1.84	3.08 3.47	40	Good
EX 39	12-17-02	78/73"	1.64 1.93	3.89 4.65	39	

Arterial Blood Gas Studies

Exhibit	**Date/Doctor**	**$_{P}CO_2$**	**$_{P}O_2$**	**Comments**
DX 46-35	1-12-95/Myers	41	69	At rest
DX 46-47	7-25-95/Dahhan	37.3 37.2	69.4 82.2	At rest After exercise
DX 46-55	6-20-96/Sargent	39	72	At rest
DX 12	1-6-00/Paranthaman	37	67	At rest
DX 26	9-6-00/Fino	40.7	63.9	At rest

DX 36	11-7-00/Rasmussen	37 38	60 65	At rest After exercise
DX 42	2-27-02/Dahhan	41.8 39.1	70.5 79.6	At rest After exercise
EX 39	12-17-02/Hippensteel	39.2 36.6	77.5 69.6	On oxygen On room air

Medical Opinion Evidence

Dr. John E. Myers Jr.

Dr. Myers examined the Claimant on January 12, 1995 (DX 46). He noted the Claimant's history of thirty five and a half years of coal mine employment, as well as his fifty year cigarette smoking habit. According to Dr. Myers, the Claimant had a positive skin test for tuberculosis in 1993, and was treated for a year with medication. His chest x-ray showed silicosis, category 1/1 p, s, in both mid and upper lung zones. Pulmonary function testing showed a mild obstructive/restrictive defect in ventilation, with the FEV1 and MVV meeting the black lung regulations criteria before the administration of bronchodilators. The arterial blood gas testing was abnormal, with hypoxemia. Dr. Myers diagnosed coal workers' pneumoconiosis, based on the x-ray; chronic obstructive pulmonary disease; and a history of a positive skin test for tuberculosis, post treatment for a year without evidence of activity.

According to Dr. Myers, the Claimant's silicosis resulted from his history of exposure to coal and rock dust. He felt that it contributed to his respiratory function impairment, and prevented arduous manual labor.

Dr. Harold L. Bushey

Dr. Bushey examined the Claimant on February 22, 1995 (DX 46-35).[3] He noted the Claimant's coal mine employment history, as well as his smoking history. The Claimant's x-ray showed emphysematous changes throughout the lung fields, with some nodes bilaterally. He classified the x-ray as 2/1, p, q. Dr. Bushey's diagnosis was chronic lung disease with pulmonary emphysema and fibrosis, compatible with pneumoconiosis. He stated that the Claimant was totally disabled.

Dr. Matt Vuskovich

[3] Although Dr. Bushey stated in his report that the date of examination was February 22, 1994, this appears to be a typographical error, as the dates on the testing results indicate it was February 22, 1995.

Dr. Vuskovich examined the Claimant on May 22, 1995 (DX 46). He noted the Claimant's history of thirty five and a half years of coal mine employment, as well as his approximately 55 year history of smoking. Pulmonary function studies showed a mild obstructive impairment both before and after bronchodilator therapy, although there was a dramatic response to the bronchodilator therapy. The x-ray showed evidence of bullae and emphysema, and hyperinflation, with evidence of old granulomatous disease. Dr. Vuskovich classified the x-ray as 0/0.

Dr. Vuskovich diagnosed chronic obstructive pulmonary disease, secondary to cigarette abuse, and mild obstructive impairment, secondary to chronic obstructive pulmonary disease. Dr. Vuskovich had earlier examined the Claimant in April 1993, and noted that his x-ray showed deterioration of the pulmonary architecture since that time. He noted that the Claimant had developed additional emphysematous changes, making it obvious that he had a disease of the bronchial tree. He felt that with cessation of smoking and proper therapy, the Claimant would experience significant improvement in his pulmonary function, and maybe even a return to normal pulmonary function.

Dr. Vuskovich found no objective evidence to support a diagnosis of coal workers' pneumoconiosis. Although the pulmonary function studies showed a mild obstructive impairment, he felt that it was in no way related to his occupation in the coal industry, but was related to his 55 year smoking history. According to Dr. Vuskovich, the Claimant could still perform his previous work in the coal industry, or work requiring similar effort.

Dr. Vuskovich provided a report dated August 25, 1995, after reviewing Dr. Dahhan's July 1995 report (DX 46-49). He stated that the spirometry results were consistent with chronic obstructive pulmonary disease secondary to cigarette abuse. He also noted that in pneumoconiosis, one would expect a diffusion barrier abnormality, but the Claimant's arterial blood gas study with exercise showed no such abnormality. According to Dr. Vuskovich, the hallmark of pulmonary disease caused by cigarette abuse is obstructive impairment. He stated that experts agree that only in the highest categories of simple coal workers' pneumoconiosis, and progressive massive fibrosis, is there any pulmonary or respiratory impairment, and even then, it is usually restrictive in nature, as opposed to the obstructive impairment caused by cigarette smoking. Dr. Vuskovich went into great detail describing the differences between restrictive and obstructive disease, stating that since pneumoconiosis is not a disease of the bronchial tree, one would expect at most a mild restrictive impairment with the higher categories of simple pneumoconiosis.

Dr. Vuskovich continued to feel that the Claimant suffered from chronic obstructive pulmonary disease secondary to cigarette abuse, or in other words, a disease of the bronchial tree. But he did not have radiographic evidence of even simple coal workers' pneumoconiosis.

Dr. A. Dahhan

Dr. Dahhan examined the Claimant on July 25, 1995 (DX 46). He noted the Claimant's

35 year history of coal mine employment, as well as his smoking history. Arterial blood gas testing was normal both at rest and after exercise. Carboxyhemoglobin levels were 7%, indicating a person who smoked over a pack a day. The spirometry testing showed a mild obstructive defect, with partial reversibility after administration of bronchodilators. The lung volume measurements showed moderate air trapping, and overall, a mild obstructive defect with no evidence of any restrictive ventilatory abnormality. The Claimant's x-ray showed hyperinflated lungs, consistent with emphysema and mild scoliosis. There were no pleural or parenchymal abnormalities consistent with pneumoconiosis.

Dr. Dahhan also reviewed medical records of the Claimant. He concluded that there were insufficient objective findings to justify a diagnosis of pneumoconiosis, based on the obstructive abnormality on clinical examination of the chest, the lack of any restrictive ventilatory abnormality on pulmonary function studies, the negative x-ray, and the normal blood gas results. He based his diagnosis of chronic obstructive lung disease on the Claimant's history of cough, sputum production and wheeze, obstructive abnormality on clinical examination of the chest, and obstructive abnormality on pulmonary function studies.

From a respiratory standpoint, Dr. Dahhan felt that the Claimant retained the capacity to return to his previous coal mining work. The cause of his chronic obstructive disease is his fifty pack years of smoking. He found no evidence of pulmonary impairment or disability caused by, contributed to, or aggravated by coal dust exposure or occupational pneumoconiosis, as his only pulmonary abnormality is obstructive in nature, with no evidence of any restrictive ventilatory defect as seen in persons with pulmonary disability secondary to coal dust exposure. As the Claimant's examination and test results indicate, his pulmonary impairment is purely obstructive, with no restrictive component, and thus is not due to coal workers' pneumoconiosis.

Dr. Dahhan examined the Claimant again on February 27, 2002 (DX 42). On examination of the Claimant's chest, he found an increased AP diameter, with hyperresonance to percussion; auscultation showed prolongation of the expiratory phase with scattered bilateral expiratory wheeze. There was no audible crepitation or pleural rubs. The Claimant's x-ray showed hyperinflated lungs, consistent with emphysema, with mild cardiac enlargement. Otherwise, the lung fields were clear, with no pleural or parenchymal abnormalities consistent with pneumoconiosis. Dr. Dahhan also performed pulmonary function and arterial blood gas studies.

Dr. Dahhan also reviewed the Claimant's medical records. He concluded that there was insufficient objective data to justify a diagnosis of coal workers' pneumoconiosis, based on the obstructive abnormalities on clinical examination, the obstructive abnormality with various response to bronchodilator therapy, the slight alteration in the blood gas exchange mechanisms that subsides with exercise, and the negative x-ray readings. Dr. Dahhan felt that the Claimant has chronic obstructive lung disease, consisting of chronic bronchitis and emphysema, and from a respiratory standpoint, is not able to perform his previous work, based on the pulmonary function study findings. But this disability is the result of his lengthy smoking habit, which has caused him to develop chronic bronchitis and emphysema.

According to Dr. Dahhan, the Claimant's pulmonary impairment was not caused by, related to, contributed to or aggravated by his inhalation of coal dust or coal workers' pneumoconiosis. Dr. Dahhan noted that the Claimant had not had exposure to coal dust since 1983, an absence sufficient to cause cessation of any industrial bronchitis. In addition, his airway obstruction showed significant response to bronchodilator therapy, which is inconsistent with the permanent adverse effects of coal dust. He also noted that the Claimant's treating physician was providing him multiple bronchodilators, indicating that he believes his condition is responsive to these measures. Finally, he noted that the Claimant takes a beta blocker, which can cause bronchospasms and airway obstruction in a susceptible individual.

Dr. Dahhan stated that the Claimant has coronary artery disease with a previous myocardial infarction, hyperlipidemia, allergic rhinitis, and peptic ulcer disease, all conditions of the general public, and not related to inhalation of coal mine dust or pneumoconiosis.

Dr. Dahhan reviewed additional records, and prepared a report dated March 14, 2003 (EX 41). He noted that Dr. Smiddy failed to mention the Claimant's smoking history, which is a significant component of any pulmonary evaluation. He also indicated that although Dr. Smiddy seemed to make a significant issue of the drop in saturation from 93% to 91%, this drop was insignificant, and was not confirmed by more accurate arterial blood gas values, which are not influenced by peripheral circulation, in particular, in an elderly individual such as the Claimant.

Based on his previous examinations of the Claimant, and his review of the medical records, Dr. Dahhan again concluded that there was no evidence of occupational pneumoconiosis or pulmonary disability secondary to coal dust exposure. The Claimant has a pulmonary disability secondary to cigarette induced lung disease, but no evidence of pulmonary impairment or disability caused by, related to, contributed to, or aggravated by his inhalation of coal dust or pneumoconiosis.

Dr. Jeffrey Dale Sargent

Dr. Sargent examined the Claimant on June 20, 1996 (DX 46-55). He noted that the arterial blood gas study results were normal for age, and a chest x-ray was negative for pneumoconiosis. There was evidence of obstructive lung disease, and a granuloma in the right middle lobe. The pulmonary function tests showed a mild obstructive impairment that improved partially after administration of bronchodilators. There was air hyperinflation, air trapping, and a mildly decreased diffusion capacity.

In Dr. Sargent's opinion, the Claimant has a non-disabling ventilatory impairment that is not due to coal workers' pneumoconiosis. According to Dr. Sargent, when coal workers' pneumoconiosis causes a ventilatory impairment it does so in the face of an x-ray that shows characteristic changes of coal workers' pneumoconiosis. In addition, coal workers' pneumoconiosis causes an irreversible, mixed obstructive and restrictive ventilatory pattern. In the Claimant's case, the obstructive pattern shown on spirometry indicated partial reversibility, and the lung volumes indicated a total lung capacity which excludes a restrictive impairment.

Thus, his physiologic abnormalities are incompatible with an impairment due to coal workers' pneumoconiosis. However, the Claimant's smoking history is of sufficient magnitude to have caused him to have developed obstructive lung disease; in fact, a partially reversible, purely obstructive ventilatory impairment, with an x-ray lacking characteristic changes of coal workers' pneumoconiosis, is exactly the type of impairment one would expect to see in cigarette smoke induced lung disease.

Dr. Sargent felt that the Claimant's impairment was very mild, and that he had the respiratory capacity to do his last job, or any job in coal mining that a normal 72 year old man could be expected to perform. In other words, his mild obstructive ventilatory impairment, due to his smoking habit, is not disabling.

Dr. Robert C. Patton

Dr. Patton examined the Claimant on August 20, 1999, for evaluation of gastroesophageal reflux disease (DX 25). Dr. Patton noted that the Claimant had a past history of tuberculosis.

Dr. Joseph F. Smiddy

The record includes treatment notes from Dr. Smiddy, the Claimant's treating physician, covering the time period July 18, 2001 to December 20, 2001 (DX 36). In his December 20, 2001 office note, Dr. Smiddy indicated that the Claimant had been followed for over 19 years for severe coal workers' pneumoconiosis. He stated that the Claimant had chronic shortness of breath, and was one hundred percent totally and permanently disabled by his coal workers' pneumoconiosis. Dr. Smiddy indicated that the Claimant had some associated obstructive lung disease, and an underlying history of arthritis and coronary artery disease. According to Dr. Smiddy, the Claimant had multiple prior x-rays that showed pneumoconiosis, and repeated pulmonary function tests that showed severe obstructive disease.

At the Claimant's October 17, 2001 visit, Dr. Smiddy indicated that the Claimant's x-ray showed micronodular change in all six lobes, consistent with his prior established diagnosis of coal workers' pneumoconiosis. He also stated that, in an x-ray taken on March 15, 2001, Dr. Larry Westerfield interpreted the film as showing a profusion of 1/1, in all six lung zones.

Dr. Smiddy sent a letter to the Claimant, dated February 3, 2000, certifying that he is totally and permanently disabled by his coal workers' pneumoconiosis, which is a direct result of his forty years of exposure to heavy quantities of coal dust (DX 27). He also indicated that the Claimant had an underlying element of bronchitis, COPD, coronary disease, BPH, arthritis, and indigestion.

The record also includes treatment notes from Dr. Smiddy for the date of April 27, 2000 (DX 27). Dr. Smiddy indicated that the Claimant was there for follow up on his bronchitis and pneumoconiosis, and that his previous x-ray showed pneumoconiosis. In a letter to the Claimant of the same date, Dr. Smiddy stated that it was his opinion that the Claimant was totally and

permanently disabled by his coal workers' pneumoconiosis.

Dr. Smiddy wrote a letter dated April 18, 2002, certifying that the Claimant was totally and permanently disabled by coal workers' pneumoconiosis (CX 5). He noted that he had done so about twenty years earlier, and that the Claimant stopped mining based on his advice. Dr. Smiddy cited to the Claimant's p02 at rest in 1982, when he was admitted to the hospital, the March 15, 2001 x-ray, which showed a profusion of 1/0, and the October 17, 2001 pulmonary function study results, which showed a rather profound obstructive defect, and moderate to severe impairment of transfer factor. He also noted that on October 17, the Claimant desaturated from 93% to 91% after walking 50 feet.

Dr. Smiddy also provided a progress note dated January 2, 2003 (CX 7). He noted that the Claimant had desaturated to 91% while walking on April 18, 2002. He indicated that the Claimant looked increasingly impaired, and there were several x-rays on file indicating the presence of coal workers' pneumoconiosis, and multiple abnormal pulmonary function test results.

Dr. S. K. Paranthaman

Dr. Paranthaman examined the Claimant on January 6, 2000 (DX 11). He administered an x-ray, which was negative for pneumoconiosis, and pulmonary function and arterial blood gas testing. His diagnosis was pulmonary emphysema with airflow obstruction, atherosclerotic heart disease with history of previous myocardial infarction, and angina pectoris. Dr. Paranthaman indicated that the Claimant's pulmonary emphysema was due primarily to his thirty pack year history of smoking, but that if forty years of coal mine employment was documented, it could have aggravated this condition. According to Dr. Paranthaman, the Claimant's respiratory impairment was moderately severe, and he would have difficulty performing his previous coal mining job. He also felt that the Claimant's angina caused moderate impairment.

Dr. Gregory Fino

Dr. Fino examined the Claimant on September 6, 2000 (DX 26). He noted the Claimant's history of coal mine employment, as well as his history of smoking. The Claimant indicated that he had shortness of breath for the last thirty years, which was getting worse. He also had dyspnea, and daily cough. The Claimant's x-ray showed no pleural or parenchymal abnormalities consistent with pneumoconiosis. Spirometry showed severe obstruction, with no bronchodilator response; the TLC was elevated, and there was air trapping. Diffusing capacity was reduced, but oxygen saturation was normal; there was mild hypoxia.

Based on his examination of the Claimant, as well as his review of the medical records, Dr. Fino concluded that the Claimant has severe chronic bronchitis and emphysema due to smoking, but that he does not suffer from an occupationally acquired pulmonary condition as a result of his coal mine dust exposure. He based this conclusion on the fact that the majority of x-ray readings, including his own, were negative. In addition, the spirometric evaluations show an obstructive

ventilatory abnormality, based on the reduction in the FEV1/FVC ration, which has occurred in the absence of any interstitial abnormality. The obstruction also shows involvement in the small airways.

According to Dr. Fino, this type of finding is not consistent with a coal dust related condition, but is consistent with conditions such as cigarette smoking, pulmonary emphysema, non occupational chronic bronchitis, and asthma. He indicated that minimal obstructive lung disease had been described in working coal miners, called industrial bronchitis. But this condition resolves within six months of leaving the mines. He acknowledged that obstructive lung disease may arise from pneumoconiosis, when there is significant fibrosis which results in the obstruction. However, in the Claimant's case, the obstruction was unrelated to his coal mine dust exposure. As the TLC was not reduced, restrictive lung disease and significant pulmonary fibrosis were ruled out.

Dr. Fino engaged in a lengthy discussion about the diagnosis of coal mine dust related lung disease, and the need for objective testing to determine impairment. He indicated that from a functional standpoint, the Claimant's pulmonary system was abnormal, and he did not retain the capacity to perform all the requirements of his last job. According to Dr. Fino, there are two risk factors for his disability, his coal mine dust exposure and his smoking. He felt that the clinical information was consistent with a smoking related disability. According to Dr. Fino:

> Even if industrial bronchitis due to coal mine employment contributed to the obstruction, the loss in the FEV1 would be in the 200 cc range. If we gave back to him that amount of FEV1, this man would still be disabled. This medical estimate of loss in FEV1 in working miners was summarized in the 1995 NIOSH document. Although a statistical drop in the FEV1 was noted in working miners, that drop was not clinically significant. This man would be as disabled had he never stepped foot in the mines.

Dr. Fino felt that it was possible to differentiate obstruction caused by coal mine dust from obstruction caused by other factors such as smoking and asthma, and he criticized a number of studies that evaluated obstruction in coal miners. He acknowledged that some miners have clinically significant obstruction as a result of coal mine dust inhalation, which would be expected in most cases of severe fibrosis where there is a combined obstructive and restrictive defect. But he felt that there was no evidence that there was a clinically significant reduction in FEV1 as a result of chronic obstructive lung disease due to coal mine dust inhalation. Nor did he feel that there was credible scientific support for the proposition that the type of emphysema that arises out of coal dust exposure is impairing or disabling, in the absence of progressive massive fibrosis.

Based on his review of the literature, Dr. Fino stated that there had been confusion about the distinction between focal emphysema and centrilobular emphysema, but regardless of this debate, clinical impairment as a result of emphysema is the gold standard in evaluating a miner's pulmonary status. He indicated that the amount of emphysema increases with the severity of pneumoconiosis; this is not true in simple silicosis. The increasing severity of simple pneumoconiosis, by x-ray or autopsy, is not correlated with a worsening of lung function. Finally,

the studies of emphysema in miners have not shown clinically significant deterioration in lung function as the emphysema worsens.

According to Dr. Fino, there has been a worsening in the Claimant's lung function since 1995, but this change is consistent with smoking, not pneumoconiosis. He concluded that there was insufficient objective medical evidence to justify a diagnosis of simple pneumoconiosis, and the Claimant does not suffer from an occupationally acquired pulmonary condition. The Claimant has a disabling respiratory impairment, which prevents him from returning to his previous mining job. Even if he assumed that the Claimant has pneumoconiosis, it does not contribute to his disability, and he would be as disabled if he never stepped foot in the mines.

Dr. Fino reviewed the Claimant's medical records at the request of the Employer, and provided a report dated October 19, 2000 (DX 30). Dr. Fino had previously reviewed the Claimant's medical records in connection with earlier claims, and examined the Claimant, and concluded that there was insufficient evidence to justify a diagnosis of simple pneumoconiosis. He did conclude that the Claimant had a disabling respiratory impairment, but it was unrelated to his inhalation of coal mine dust. Finally, he noted a worsening of the Claimant's lung function since 1995, a change that was consistent with smoking.

Dr. Fino reviewed the pulmonary function study performed on August 30, 1999, finding that the values showed moderately severe obstructive lung disease. He indicated that the MVV was invalid, and underestimated the Claimant's true lung function. He also reviewed the pulmonary function study performed on January 17, 2000, indicating that it showed moderately severe obstructive lung disease, with improvement after bronchodilators. Based on his review of additional medical records, Dr. Fino did not change any of his previous opinions.

Dr. D. L. Rasmussen

Dr. Rasmussen examined the Claimant on November 7, 2000 (DX 36). He noted the Claimant's symptoms of progressive shortness of breath with exertion, significant dyspnea after climbing stairs, and chronic productive cough. He also noted the Claimant's history of coal mine employment, and his smoking history. On physical examination of the Claimant, Dr. Rasmussen noted moderately reduced breath sounds, and bilateral basilar rales. There was a prolonged expiratory phase with forced respirations. A chest x-ray read by Dr. Patel showed pneumoconiosis 1/1, s, s. The pulmonary function studies revealed moderate, slightly reversible obstructive insufficiency, with the maximum breathing capacity moderately reduced. The single breath carbon monoxide diffusing capacity was moderately to markedly reduced, and there was minimal resting hypoxia. On the treadmill study, the Claimant's volume of ventilation was markedly increased, and he retained a breathing reserve of only 13 L/min., indicating limitation due to ventilatory impairment. There was a significant increase in the VD/VT ratio, and distinct increase in arterial to end tidal pc02 of 7.5. There was moderate impairment in oxygen transfer.

Dr. Rasmussen concluded that, overall, the studies indicated at least moderately severe loss of respiratory function, as reflected by the ventilatory impairment, the reduced diffusing

capacity, the impairment in oxygen transfer, and the increased dead space ventilation during exercise. According to Dr. Rasmussen, this degree of impairment would make the Claimant totally disabled for performing more than sedentary work, and he is clearly disabled for resuming his former coal mining work, which required heavy manual labor.

Dr. Rasmussen noted the Claimant's long history of exposure to coal mine dust, and x-ray changes consistent with pneumoconiosis; he stated that it is medically reasonable to conclude that the Claimant has coalworkers' pneumoconiosis, which arose from his coal mine employment. He indicated that the two risk factors for the Claimant's disabling respiratory insufficiency are his cigarette smoking and his coal mine dust exposure, with the coal mine dust exposure being a major contributing factor to his disabling respiratory insufficiency.

Dr. Kirk E. Hippensteel

Dr. Hippensteel examined the Claimant on December 17, 2002 (EX 39). He noted his history of coal mine employment, as well as his history of cigarette smoking. The Claimant indicated that he had had breathing problems for thirty years. His x-rays showed a minimal increase in irregular markings, consistent with classification of 0/1, s, t. Pulmonary function testing suggested moderate obstruction with 18% improvement in the FEV1 after bronchodilators, or up to the mild range of impairment. According to Dr. Hippensteel, this amount of reversibility is suggestive of asthma. The Claimant's MVV was severely reduced, with suboptimal tidal volumes; his lung volumes showed air trapping and no evidence of restriction, and his diffusion was severely reduced. The Claimant's arterial blood gas study was normal for age with oxygen removed.

Based on this data, Dr. Hippensteel did not feel that a diagnosis of coal workers' pneumoconiosis was justified. He noted that the Claimant had partially reversible airflow obstruction, possibly aggravated by a recent bronchial infection, but enough reversibility to suggest asthma. After administration of bronchodilators, he felt that the Claimant had good enough function from a pulmonary standpoint to work at his previous job, even though he has some impairment from smoking and scoliosis. However, he is impaired as a whole man due to age, heart disease, hypertension, abdominal aortic aneurysm, and allergies.

Dr. Hippensteel also reviewed the Claimant's previous medical records, noting that over 80% of the x-ray interpretations were negative for pneumoconiosis. Even though the Claimant's pulmonary function studies worsened in the previous three years, at times he had significant reversibility, which is indicative of bronchial inflammation that fits with episodes of recurrent bronchitis and asthmatic features associated with his allergy history, and not pneumoconiosis. He noted that even though Dr. Rasmussen's November 2000 arterial blood gas study met the disability criteria, the gas exchange improved significantly in February 2002 as well as in Dr. Hippensteel's study, showing that this impairment was not permanent, and therefore not related to coal workers' pneumoconiosis.

Based on his review of the medical reports, and his examination of the Claimant, Dr.

Hippensteel concluded that the Claimant has evidence of a variable, partially reversible obstructive respiratory impairment, but it is not typical or suggestive of pneumoconiosis as a cause. For the most part, his x-rays have been negative. According to Dr. Hippensteel, it would not be typical with pneumoconiosis to have variable interstitial findings from one year to the next, since if pneumoconiosis develops radiographically, it is permanent or progressive. He noted that the Claimant had a history of allergies, and even though he denied having asthma, one physician had diagnosed it. It is not uncommon for persons with allergic rhinitis to have airway reactivity problems also.

Looking at the characteristics of the Claimant's impairment, as well as the findings in total, Dr. Hippensteel indicated that, at times the Claimant's impairment has been severe enough to disable him from a pulmonary standpoint, but at times the impairment has not been so severe. Thus, the Claimant does not have a permanent change in pulmonary function which keeps him from working in the coal mines. He is impaired as a whole man from a combination of problems, including his age, but pneumoconiosis has not been the cause, despite his long history of coal mine dust exposure. His other risk factors for pulmonary disease have created his chronic bronchitis, and likely worsened his function.

Dr. Hippensteel reviewed the Claimant's medical records, and prepared a report dated February 24, 2003 (EX 40). He noted that Dr. Smiddy reported many subjective complaints by the Claimant, but only documented one pulmonary function study indicative of severe obstruction, without any comments about reversibility except for the fact that he was treating the Claimant with bronchodilator medication. Dr. Hippensteel felt that Dr. Smiddy overstated impairment on some of the tests, and that there was some disconnection between his claim that his only diagnosis was coal workers' pneumoconiosis, and his own findings. According to Dr. Hippensteel, the lack of inclusion of other evidence of reversibility and improved function indicated that Dr. Smiddy was not looking objectively at the evidence about causation and impairment. In particular, he felt that it was a gross oversight to ignore the Claimant's more than fifty pack year history of smoking as a cause for impairment.

Dr. Hippensteel stated that the results of the August 1, 2002 spirometry test showed wide variation in FEV1, indicative of inconstant effort, and therefore underestimated the Claimant's true function.

Dr. Hippensteel concluded that the Claimant had not developed pneumoconiosis, or enough permanent impairment in pulmonary function to prevent him from working at his former coal mine job. He based this on the reversibility on various pulmonary function tests, which is not typical for pneumoconiosis, which causes a fixed, permanent or progressive impairment. The majority of x-ray readings were negative, including the most recent x-ray. Dr. Hippensteel felt that the Claimant was impaired as a whole man from going back to his previous job.

DISCUSSION

Judge Kaplan accurately summarized the prior evidence of record, finding that the newly submitted evidence in that proceeding was insufficient to establish that the Claimant had pneumoconiosis, or that he had a totally disabling respiratory impairment. Similarly, Judge McElroy determined that the evidence before him was insufficient to establish pneumoconiosis or a totally disabling respiratory impairment. Thus, in order to establish a material change in conditions, and thus entitlement to consideration of his claim on the merits, the Claimant must establish by a preponderance of the evidence either that he has pneumoconiosis, or that he has a totally disabling respiratory impairment.

I find that the newly submitted evidence clearly establishes that the Claimant has a totally disabling respiratory impairment. Although the new arterial blood gas study results, with one exception before the administration of bronchodilators, do not meet the regulatory presumption of total disability, the majority of the more recent newly submitted pulmonary function study results do.[4]

In addition, the physicians who have examined the Claimant most recently have concluded that he now has a totally disabling respiratory impairment.[5] Thus, Dr. Dahhan, who examined the Claimant in 1995 and concluded that he retained the respiratory capacity to return to his former coal mining work, determined, after examining him in 2002, that the Claimant has chronic obstructive lung disease, in the form of chronic bronchitis and emphysema, and is not able to perform his previous work.

Dr. Smiddy, the Claimant's treating physician, has indicated that the Claimant has a totally disabling respiratory impairment, and this is consistent with the objective medical evidence of record.

Dr. Paranthaman, who examined the Claimant in January 2000, concluded, based on his examination and testing, that he had pulmonary emphysema with airflow obstruction, due primarily to his history of smoking, which would cause him difficulty in performing his previous coal mining job. Dr. Rasmussen also examined the Claimant, in November 2000, and concluded, based on pulmonary and arterial blood gas tests, as well as his examination of the Claimant, that he had at least a moderately severe loss of respiratory function, which disabled him from resuming his previous coal mining work.

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[4] The tables at Appendix B do not cover persons past the age of 71. The Claimant's results, at any of the heights reported, are below the values for a person of age 71. As the values in the table steadily decrease with age, it is reasonable to assume that if anything, the values for a person past the age of 71 would also decrease; they certainly would not be greater.

[5] Dr. Bushey, who examined the Claimant in February 1995, also concluded that the Claimant was totally disabled, although it is not clear what his basis for this conclusion was, since the Claimant's pulmonary function results were above disability standards.

Based on the Claimant's spirometry results, his examination of the Claimant, and his review of the medical records, Dr. Fino concluded that the Claimant has severe chronic bronchitis and emphysema due to his smoking, which prevents him from returning to his previous coal mining job.

On the other hand, Dr. Hippensteel, who examined the Claimant in December 2002, concluded that although the Claimant had obstructive impairment, after administration of bronchodilators, his pulmonary function was good enough for him to be able to work at his previous job.

I find that the consistent reports of Dr. Dahhan, Dr. Smiddy, Dr. Rasmussen, and Dr. Fino, as well as the recent qualifying pulmonary function study results, outweigh Dr. Hippensteel's determination that, with the administration of bronchodilators, the Claimant can perform his previous job from a respiratory standpoint. Thus, I find that the Claimant has established that he has a totally disabling respiratory impairment pursuant to Section 718.204(b)(2)(i) and (iii). Therefore, the Claimant is entitled to consideration of his claim on the merits.

Merits of the Claim

To be entitled to benefits, the Claimant must establish that he has pneumoconiosis, and that his total respiratory disability is due to pneumoconiosis.

According to 20 C.F.R. §718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (§718.202 (a)(1)), autopsy or biopsy report (§718.202 (a)(2)), regulatory presumption (§718.202 (a)(3)), and physician medical opinion (§718.202 (a)(4)). Because the record does not contain any evidence of complicated pneumoconiosis, and the Claimant filed his current claim after January 1, 1982, the regulatory presumption of pneumoconiosis is not applicable. In addition, the official record does not contain an autopsy report or a biopsy report. As a result, Claimant must rely on chest x-ray evidence or medical opinion to establish the existence of pneumoconiosis.

The eighty one x-ray interpretations submitted in connection with the proceeding before Judge McElroy were predominantly negative, as were the thirty four interpretations reviewed by Judge Kaplan. The newly submitted evidence includes seventy-one interpretations of seventeen x-rays performed since Judge Kaplan's February 1994 denial. The overwhelming majority of these interpretations, almost all of them by dually qualified readers, are negative for pneumoconiosis. Thus, the January 12, 1995 x-ray was read as positive by Dr. Bassali, who is dually qualified, and Dr. Myers, whose qualifications are unknown. However, Dr. Wiot, Dr. Spitz, Dr. Scott, and Dr. Wheeler, all dually qualified, and Dr. Sargent and Dr. Fino, B readers, read this x-ray as negative. Given the preponderance of negative readings by the physicians with the highest qualifications, I find that this particular x-ray is negative for pneumoconiosis.

The next x-ray, performed on February 22, 1995, was again read as positive by Dr. Bassali

and Dr. Bushey, but as negative by Dr. Wiot, Dr. Barrett, and Dr. Spitz, all dually qualified, and Dr. Sargent and Dr. Fino, B readers, as negative. Again, given the preponderance of negative readings by the most highly qualified readers, I find that this x-ray is negative for pneumoconiosis.

There are twenty three interpretations of the next six x-rays, performed on May 15, 1995, May 22, 1995, July 25, 1995, June 20, 1996, January 6, 2000, and January 27, 2000, the majority of them by dually qualified physicians. The interpretations are uniformly negative.

The x-ray performed on February 3, 2000 was interpreted by Dr. Alexander, who is dually qualified, as positive for pneumoconiosis. However, Dr. Wheeler, Dr. Scott, Dr. Barrett, and Dr. Ramakrishnan, all dually qualified, and Dr. Fino, a B reader, interpreted this x-ray as negative. Given the preponderance of negative interpretations by the most highly qualified readers, I find that this x-ray is negative.

There are eight interpretations of the next two x-rays, performed on May 8, 2000, and September 6, 2000, mostly by dually qualified readers. These readings are uniformly negative.

The x-ray dated November 7, 2000 was interpreted by Dr. Patel, a dually qualified reader, as positive. However, Dr. Scott and Dr. Scatarige, who are dually qualified, and Dr. West, who is a B reader, interpreted it as negative. Given the preponderance of negative readings by the most highly qualified physicians, I find that this particular x-ray is negative.

There are five interpretations of the next two x-rays, performed on December 26, 2000, and March 15, 2001, all but one by dually qualified readers. They are uniformly negative.

Dr. Smiddy provided an interpretation of the October 17, 2001 x-ray, finding that it showed micronodular changes consistent with pneumoconiosis; he did not complete an ILO form, or classify the x-ray by ILO standards. However, Dr. Barrett, Dr. Scatarige, and Dr. Wheeler, all dually qualified, read this x-ray as negative. Given the qualifications of these readers, I find that this x-ray is negative for pneumoconiosis.

There are seven interpretations of the last two x-rays, performed on February 27, 2002, and December 17, 2002, three by B readers, and four by dually qualified readers. These interpretations are all negative.

I find that the overwhelming preponderance of the x-ray interpretations, both new and old, by physicians with the highest qualifications, is negative for pneumoconiosis, and therefore the Claimant has not established the existence of pneumoconiosis by x-ray evidence.

Judge Kaplan relied on the reports by Dr. Dahhan, Dr. Vuskovich, and Dr. Fino to conclude that the Claimant had not established the existence of pneumoconiosis by medical opinion evidence. The other two physicians, Dr. Baker and Dr. Vaezy, based their diagnoses of pneumoconiosis largely on the Claimant's x-ray, which I have found does not establish the existence of pneumoconiosis. Judge McElroy found that the opinion by Dr. Neill, that the

Claimant's chronic bronchitis was the result of his history of smoking, but that he did not have pneumoconiosis, outweighed the opinion by Dr. Kanwal, who found pneumoconiosis on the basis of an x-ray interpretation, and the inconsistent reports by Dr. Smiddy. Both Judge Kaplan and Judge McElroy accurately summarized the medical opinion evidence, and for the same reasons, I find that the medical opinion evidence submitted in connection with the previous claims does not establish the existence of pneumoconiosis.

The newly submitted medical evidence includes a report by Dr. Myers, who examined the Claimant in January 1995, and diagnosed coal workers' pneumoconiosis. However, he based this determination on his positive reading of the Claimant's x-ray, while I have found that the x-ray evidence is overwhelmingly negative for pneumoconiosis. Thus, I accord little weight to Dr. Myers' conclusion.

Similarly, Dr. Bushey, who examined the Claimant in February 1995, diagnosed the Claimant with pneumoconiosis, based on his interpretation of the Claimant's x-ray. Although Dr. Bassali also found this x-ray to be positive, five other physicians, three of them dually qualified, found it to be negative. Additionally, the overwhelming majority of x-ray interpretations since that time have been negative. Thus, I place little weight on Dr. Bushey's conclusions.

Dr. Rasmussen, who examined the Claimant in November 2000, concluded that it was "medically reasonable" to find that he has pneumoconiosis, based on his long history of exposure to coal mine dust, and his x-ray changes consistent with pneumoconiosis. Again, I have found that the overwhelming preponderance of the newly submitted x-ray evidence is negative for pneumoconiosis. I find that Dr. Rasmussen has failed to offer a sufficient basis for his conclusions, and that his opinion is not sufficient to establish that the Claimant has pneumoconiosis. *See, Cornett v. Benham Coal*, 227 F.3d 569 (6th Cir. 2000).

Dr. Smiddy is the Claimant's treating physician, and he has stated that he has treated the Claimant for about twenty years for pneumoconiosis. Although he referred to "multiple" prior x-rays that showed pneumoconiosis, the only x-rays he specifically identified were the October 17, 2001 x-ray, which Dr. Smiddy felt showed micronodular change in all six lobes, consistent with the Claimant's previous diagnosis of pneumoconiosis, and the x-ray of March 15, 2001, which he claimed was interpreted by Dr. Larry Westerfield as showing a profusion of 1/1, in all six lung zones. Dr. Smiddy did not complete an ILO form for his interpretation of the October 17, 2001 film, or otherwise classify it according to ILO standards. There is no interpretation by Dr. Westerfield of the March 15, 2001 x-ray in the record.[6] In any event, both of these x-rays, as well as many before and after, were interpreted as negative by numerous dually qualified physicians. As this appears to be the only basis for Dr. Smiddy's diagnosis of pneumoconiosis, I find that it is not supported by the objective medical evidence of record, and it is entitled to little weight.

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[6] There are two interpretations by Dr. Westerfield, of the September 30, 1982 x-ray (DX 45-13), and the July 7, 1983 x-ray (DX 45-41), both of which are negative for pneumoconiosis.

Dr. Vuskovich examined the Claimant and reviewed his medical records in 1995, finding no objective evidence to support a diagnosis of pneumoconiosis. I find that, while his reports are well-reasoned, they do not provide any information about whether the Claimant developed pneumoconiosis since that time, and thus are of very limited value.

Dr. Dahhan also examined the Claimant and reviewed his medical records in 1995, finding insufficient objective evidence of pneumoconiosis. Dr. Dahhan examined the Claimant again in February 2002, and reviewed his medical records as late as March 2003, again finding no evidence of pneumoconiosis. He based his conclusion on the negative x-ray readings, the significant reversibility of the Claimant's airway obstruction with bronchodilator therapy, which is inconsistent with the permanent adverse effects of coal dust, and the slight alteration in blood gas exchange mechanisms that subsides with exercise. Dr. Dahhan also relied on the fact that the Claimant's impairment is purely obstructive, with no restrictive abnormality; in other words, having concluded that the Claimant has an obstructive impairment, Dr. Dahhan concluded that he does not have pneumoconiosis, since he suffers a type of impairment (*i.e.*, obstructive disease) which Dr. Dahhan believes pneumoconiosis cannot produce. This view, which is clearly contrary to the Court's decision in *Warth v. Southern Ohio Co.*, 60 F.3d 173 (4th Cir. 1995), as clarified by *Stiltner v. Island Creek Coal Co.*, 86 F.3d 337 (4th Cir. 1996), causes me to place somewhat less reliance on Dr. Dahhan's otherwise well-reasoned opinions.

Dr. Sargent examined the Claimant in 1996, noting that his chest x-ray was negative. Although the Claimant's pulmonary function tests showed a mild obstructive impairment, Dr. Sargent felt that it was not due to pneumoconiosis, which causes impairment only when there is a positive x-ray. He also relied on the fact that pneumoconiosis causes an irreversible, mixed obstructive and restrictive ventilatory pattern. As spirometry testing showed partial reversibility, and lung volumes excluded a restrictive impairment, he concluded that the physiologic abnormalities were incompatible with impairment attributable to pneumoconiosis. Again, Dr. Sargent's view that pneumoconiosis cannot cause strictly obstructive impairment, and that pneumoconiosis can result in impairment only with a positive x-ray, ignores the concept of legal pneumoconiosis as set out by the Courts. I also note that Dr. Sargent's conclusions do not address the question of whether the Claimant has established that he has pneumoconiosis in the seven years since Dr. Sargent's report. For all of these reasons, I do not accord significant weight to Dr. Sargent's opinions.

Dr. Fino examined the Claimant and reviewed his medical records in September and October 2000. He concluded that the Claimant does not suffer from an occupationally acquired pulmonary condition as a result of his coal mine dust exposure, based on the majority of negative x-ray readings, the obstructive ventilatory abnormality with no interstitial abnormality, and involvement in the small airways. He acknowledged that pneumoconiosis can produce obstructive disease, if there is significant fibrosis; but this was not the case with the Claimant.

Dr. Paranthaman, who examined the Claimant in January 2000, diagnosed pulmonary emphysema with airflow obstruction, due primarily to his thirty pack year history of smoking. He did state that if forty years of coal mine employment were documented, this "could have"

aggravated the Claimant's condition. I find this conclusion to be equivocal and speculative, and not sufficient to establish the existence of pneumoconiosis.

Finally, Dr. Hippensteel, who reviewed the Claimant's medical records in February 2003, concluded that he had not developed pneumoconiosis, based on the majority of negative x-ray readings, as well as the reversibility shown on pulmonary function tests, which is not typical for pneumoconiosis, which causes a fixed, permanent or progressive impairment. Dr. Hippensteel had the advantage of reviewing all of the Claimant's medical records, and his conclusions are based on objective medical evidence. I find that his conclusions are well-reasoned and documented, and I accord them significant weight.

While Dr. Smiddy has been the Claimant's treating physician for many years, that status alone does not entitle his opinions to determinative weight. As noted above, Dr. Smiddy relied on positive interpretations of the x-ray evidence, when the overwhelming preponderance of the interpretations are to the contrary. In addition, reading Dr. Smiddy's notes and reports as a whole, it appears that his diagnosis of pneumoconiosis is based in large part on the fact that the Claimant, who worked in the mines for many years, has respiratory problems. But nowhere does Dr. Smiddy even mention, much less discuss, the extensive smoking history of his patient. I agree with Dr. Hippensteel, that this suggests a lack of objectivity on Dr. Smiddy's part. Dr. Smiddy's opinions are not consistent with the objective medical evidence of record, and he offers no rationale to support his opinions, other than positive x-ray readings, the Claimant's history of coal mine employment, and the fact of impairment, while ignoring the Claimant's very significant smoking history.

It is the Claimant's burden to establish the existence of pneumoconiosis by a preponderance of the medical evidence. For the reasons discussed above, I find that the opinions of Dr. Smiddy, Dr. Rasmussen, Dr. Bushey, and Dr. Myers are insufficient to establish the existence of pneumoconiosis, and they are outweighed by the well-reasoned and supported opinions of Dr. Hippensteel and Dr. Fino. Thus, I find that the Claimant has failed to establish the existence of pneumoconiosis by a preponderance of the medical opinion evidence.

I have reviewed all of the medical evidence as a whole, and I find that it does not establish the existence of pneumoconiosis. *See, Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000).

Finally, even if I were to find that the Claimant has established the existence of pneumoconiosis, I find that he has not established that his total respiratory disability is due to pneumoconiosis. For the reasons discussed above, I find the reports of Dr. Fino and Dr. Hippensteel, who concluded that the Claimant's disabling chronic bronchitis and emphysema are the result of his extensive smoking history, to be most persuasive.

CONCLUSION

The Claimant has established that he has a totally disabling respiratory impairment. But he has not established that he has pneumoconiosis, or that his total respiratory disability is due to pneumoconiosis. Therefore, he is not entitled to benefits under the Act.

ORDER

IT IS HEREBY ORDERED that the claim of Edd Logan for benefits under the Act is DENIED.

A

LINDA S. CHAPMAN
Administrative Law Judge

ATTORNEY'S FEES

The award of an attorney's fee is permitted only in cases in which the Claimant is found to be entitled to benefits under the Act. Since benefits are not awarded in this case, the Act prohibits the charging of any attorney's fee to the Claimant for legal services rendered in pursuit of this claim.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this decision, by filing a Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. *A copy of a Notice of Appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, NW, Room N-2117, Washington, D.C. 20210.*